CHAPTER 5

MODELLING HEALTH IMPACTS OF AIR POLLUTION AND THEIR VALUATION: AN APPLICATION TO SANTIAGO, CHILE

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1. INTRODUCTION

This chapter describes how estimates of air pollution emissions are transformed into ambient concentration of pollution, further translated into exposure and health impacts on a population, and finally expressed in damages. Different trade and environmental policy menus lead to different levels of pollution emissions and income. For each menu the emissions levels translate into specific mortality and morbidity impacts on the population. The impacts have different values because health risks and income levels are specific to the policy menu. The willingness to pay (WTP) to avoid or reduce air pollution increases with increasing wealth and pollution levels. We go through these processes—from emissions to valuation of mortality and morbidity impacts—for the case study of the Chilean economy described in Chapter 6, with a focus on air pollution in Santiago.

The emissions at the national level coming from the economy-wide model are calibrated to reproduce existing data on emissions of air pollution in Santiago. Then, a simple dispersion model is constructed using information on pollution concentration and emission inventory for Santiago. Dispersion modelling allows us to link emissions of effluents to ambient concentration of the effluents in the air. Once concentrations are estimated, they are applied to dose-response functions, which express the changes in incidence of morbidity and mortality induced by changes in pollution concentrations. Morbidity focuses on specific nonfatal illnesses or symptoms caused by air pollution, whereas mortality focuses on changes in the risk of death. The air pollutants of interest are small particulates, sulphur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), ozone (O_3), and lead. Finally, the health incidences are applied to Santiago's population to obtain the health impact in Santiago corresponding to national emissions estimated by the model (number of deaths, and morbidity measures, such as number of cases of bronchitis).

The last step is to value the health impacts. We describe the WTP approach to value morbidity and loss of life due to a change in risk of mortality, which was adopted by the research program. Then, we explain how we approximate the WTP for reduced morbidity and mortality in Santiago. There exists a large body of information and data on such WTP measures for industrialised economies, whereas virtually none is available for developing economies.[2] This raises a set of issues about the transferability and application of this information to a developing economy. The available measures on mortality are based on the value of a statistical life, which indicates the ex ante aggregate WTP to reduce mortality. The most widely used information comes from wage-risk studies in labour markets.

Using the available wage-risk studies, we follow and expand Bowland and Beghin's (2001) meta-analytic regression models to link the valuation of a statistical life to risk levels and to economic and demographic determinants, such as income, education, and age. We then use the estimated model to predict the value of a statistical life in Santiago as determined by the population's age, education, and income characteristics. The available studies are mostly from OECD countries, which are high-income economies. Income is an important determinant of the value of a statistical life.

Income raises some important issues. First, income should be compared internationally, accounting for purchasing power parity (PPP) across countries. Second, the fact that income levels are systematically lower in developing economies than in industrialised countries implies that predictions of the value of life in developing economies are less precise than predictions made within the income range corresponding to the industrialised countries included in the data. The technique used in the econometric estimation minimises this loss of accuracy by checking the ability of the model to predict the statistical value of a life for the data points with the lowest income and risk levels. We use various functional forms and econometric estimation methods to characterise the response of the WTP measures to reduce mortality to changes in income and risk. We find that WTP is elastic with respect to income, with a median elasticity of around 1.8, and it is inelastic with respect to risk, with a median elasticity of around 0.25. The results are very robust.

The transfer of WTP measures for morbidity is less sophisticated, because of the limited number of estimates available per measure of morbidity (for example, the value of avoiding headache or chest pain). Hence, the procedure followed for mortality is ruled out. We adapt the available estimates from industrialised countries to our study by scaling them down to reflect the per capita income difference between these countries and Chile.

In our application to air pollution in Santiago, we find that both mortality and morbidity valuations are important. We also compare the net welfare gains from growth under the various policy scenarios approximated by real aggregate income minus health damages in Santiago (the sum of the valuations of the mor-

tality and morbidity incidences of the scenarios). Under the free-trade scenario, we find that the welfare loss due to air pollution in Santiago represents 16 per cent of the real income gain induced by the reform. When an effluent tax is applied to reduce PM-10 emissions (i.e., small particulate matter with a size smaller than 10 micrograms) by 25 per cent, health damages are reduced by 17 per cent compared to damages under the business-as-usual (BAU) scenario. Overall, the effluent tax on PM-10 induces a clear net welfare gain (loss of gross domestic product plus reduction in damages).[3]

In the next section we describe the air pollution emissions included in the model. Then in Section 3 we explain how we obtain the ambient pollution concentrations. The health dose-response functions are presented in Section 4. We follow in Section 5 with the valuations of the health effects and the methodological issues involved with the estimates. Then in Section 6 we illustrate the valuation exercise with an application of the approach to the subset of the policy reforms considered in Chile. We offer brief conclusions in the final section.

2. AIR POLLUTION EMISSIONS

Here, we review the series of air pollutants included in the economy-wide model that are relevant for gauging the health impact of urban air pollution.

2.1 Total Suspended Particulates

The first air pollutant of interest is particulate matter (PART) and its subset of smaller particulates, PM-10. Particulates is a term used to describe dispersed airborne solid and liquid particles. Primary health-related concerns for suspended particulates focuses on PM-10 and not PART (Ostro 1994; Ostro et al. 1995). PM-10 is the fraction of PART made up of particulate matter smaller than 10 micrograms (μg) in size. The usual assumption to translate PART into PM-10 is to assume that PM-10 constitutes 55 per cent of PART. The computable general equilibrium (CGE) model estimates the annual national emissions level for 1992 at 43,109 metric tons of PART. This estimate is the sum of emissions in production and consumption (29,495.2 tons and 13,613.9 tons, respectively).

The majority of PART emissions in Santiago is caused by street dust blown from unpaved roads and eroded land. Industrial processes, which contribute to the particulates problem, are industrial combustion and thermal processes. These include industrial boilers for steam production, furnaces for smelting, production of ferro-alloys, drying and carbonisation processes; and heat generation using solid, liquid, and gaseous fuels in smaller boilers in buildings (The World Bank 1994; O'Ryan 1993). Street dust and wood burning contribute to about 74 per cent of PART; transportation to 11 per cent; and industry to 15 per cent.

To calibrate the PART emission data generated by the CGE model to the existing pollution inventory for Santiago (The World Bank 1994), we multiply the PART emissions of each industry/sector by an adjustment coefficient reflecting the share of that industry located in the Santiago Metropolitan Region. We use Santiago's share of total labour employed by the industries/sectors. This step is complicated by the lack of disaggregated data on industrial labour. When data is not available we use the average labour share for aggregate industrial activity. Most of the labour data comes from the 1992 census published by Banco Central de Chile (1995).

Next, emissions from final consumption are first normalised by a constant (0.925), which represents the urban share of labour income in total labour income. The urban emissions from final consumption are then multiplied by Santiago's share of total urban population. We add the two "best-guess" estimates of particulates emissions released in production and final consumption in Santiago to obtain a sum of total emissions for Santiago estimated by our model. Then, we compare the total to the existing figures coming from Santiago's emission inventory constructed by Ulriksen et al. (1994) and Turner, Weaver and Reale (1993) for The World Bank, and we rescale our "best-guess" estimates of production- and consumption-induced pollution such that their sum matches the inventory figure of PART (PM-10). We follow a similar calibration procedure for the remaining set of air effluents considered in the study.

From the existing pollution emission inventory of The World Bank (1994), the average annual emissions in Santiago are 25,423 tons of PM-10 or 46,224 tons of PART for 1992. The CGE model tends to underpredict emissions for some pollutants. This downward bias comes principally from the emission intensity estimates (see Chapter 4 on input-based estimates), which are based on U.S. data. U.S. emission intensities tend to be lower than the intensities in developing economies because of environmental regulation and technological advances. Another source of downward bias comes from not accounting for human activities (and associated pollution) that are not included in national accounts.

2.2 Sulphur Dioxide

The second type of air pollutant of interest for health effects is SO_2. From our model, the computed SO_2 emissions for Chile in 1992 are 241,429 metric tons per year. In terms of high effluent intensities, the principal sectoral sources are transportation sectors, gas, pottery, glass, non-metallic minerals, copper mining, agriculture, and agricultural services. The SO_2 emissions estimate from Santiago's inventory is 20,338 tons per year for 1992 (The World Bank 1994). The SO_2 emission figure for Santiago appears to be low, given the

long-distance transport potential of SO_2 and the presence of copper mines near Santiago. The average annual ambient concentration level is 61.46 micrograms per cubic metre ($\mu g/m^3$) for 1992 (The World Bank 1994). For Santiago specifically, primary sources are industry (71 per cent) (which excludes copper mines), transportation (24 per cent), and the rest from residential sources and offices.

2.3 Nitrogen Dioxide

Nationally, the major sources of NO_2 in Chile are similar to SO_2 sources. The two pollutant types are correlated almost perfectly. The national emission level predicted by our model is 146,951 metric tons per year for 1992. For Santiago, NO_2 emissions come from transportation (85 per cent), industry (14 per cent), street dust, and wood burning. These emissions are estimated at 25,140 tons per year in the Santiago inventory for 1992.

2.4 Carbon Monoxide

The fourth air pollutant of concern in Santiago is CO. Nationally, our model predicts annual emissions of 51,519.1 metric tons for 1992. The average annual emissions for Santiago are 291,440 tons per year for 1990-92 (The World Bank 1994). The CGE model seriously underpredicts the emissions of CO because the U.S. database used to generate our estimates of sectoral intensities reflects the major CO abatements that have occurred over the last twenty years in the United States. High emission intensities occur in coal, sugar, non-metallic minerals, basic metals, electricity, and gas. In levels, transportation sectors also contribute, although they have lower intensities than the former sectors. Primary sources of emissions for Santiago include transportation (94 per cent); the remainder comes from industrial sources (The World Bank 1994).

2.5 Volatile Organic Compounds

Volatile Organic Compounds (VOCs) do not have known health effects by themselves, but with NO_2 they are precursors of O_3, which is detrimental to health. Our model predicts 43,110.5 metric tons of annual emissions of VOCs for Chile in 1992. It underpredicts the national levels since the emission inventory in Santiago is slightly higher than the predicted figure. The average annual emissions level for Santiago is 47,918 tons per year (The World Bank 1994). Nationally, the sectors with high concentrations of VOCs are wine and liquor, furniture, chemical industries, and petroleum refining. In terms of volume, construction is another significant contributor. The primary sources of emissions for Santiago are transportation (83 per cent), sector for home and automotive-

painting small businesses (13 per cent), and residential (4 per cent). In our model, transportation is an important intermediate consumption source for many sectors, and emissions related to transportation come from all these sectors.

2.6 Lead

We assume that lead emissions constitute a fixed per centage of bio-accumulative effluents released in the air. The major sources of bio-accumulative air pollution are metallic industries, mining, electrical machinery, transport material, and electricity. The calibration of national emissions to emissions in Santiago follows the same adjustment procedure as for the other pollutants. The major source of lead pollution in Santiago is transportation, due to leaded gasoline consumption. Inasmuch as the United States phased out leaded gas in the 1980s, our effluent intensity estimates are again biased downward.

3. DISPERSION

Dispersion modelling maps the effluent emissions into ambient concentration levels. The process takes into account spatial distribution of the pollutant (e.g., distance and direction), temporal aspect (e.g., length of time in the air), and geographical population densities. Population-weighted concentration levels are used later to determine exposure rates for health impacts. Dispersion of air pollutants depends on many parameters such as the type of pollutant considered, meteorological conditions, topology, climate, and technology. Computer models generating the dispersion and resulting concentrations of pollutants are expensive and complex.

We assume linear dispersion of emissions. For most pollutants we rely on information generated by Ulriksen et al. (1994) for The World Bank, which provides emissions levels in Santiago for base and control scenarios and the corresponding ambient concentrations, using a dispersion model. As a special case, for the determination of O_3 concentration we use information from the National Research Council, the Office of Technology Assessment of the U.S. Congress, and Radian to link VOCs and NO_2 emissions to concentrations of O_3. Despite their simple nature, The World Bank estimates do account for both population and location. The linearisation of emission dispersion is a reasonable approximation if the following conditions hold:

1. If Santiago's climatology remains the same. The topo-climatology of Santiago basically dampens the dispersion of air pollutants in Santiago. Santiago is surrounded by the Andes Mountains, and is typified by anticyclonic conditions, which limit the transport of air pollutants into or out of the region (Sanchez 1992).
2. If pollutants evolve in similar proportion to those currently observed. Our emission estimates for the policy scenarios do not vary much outside the range of The World Bank estimates.

The following describe the concentrations and dispersion equations used to compute the concentrations, for each of the air pollutants considered. Next, we consider concentrations of PM-10, SO_2, NO_2, CO, O_3, and lead.

PART and PM-10. The average annual ambient level of PM-10 is estimated at 112.603 μg/m^3 (about 205 μg for PART) for Santiago in 1991 (The World Bank 1994). The dispersion equation for PM-10 is as follows:

$$PM\text{-}10 \text{ in } mg/m^3 = 56.6675 + 0.000220047 \text{ (PM-10 in metric tons/year).} \quad (1)$$

SO_2. The SO_2 dispersion equation is as follows:

$$SO_2 \text{ in } mg/m^3 = 2.7954 + 1.6476\ 10\text{-}4 \text{ (SO_2 in metric tons/year).} \quad (2)$$

NO_2. We use the following equation to link NO_2 emissions and concentration:

$$NO_2 \text{ in pphm} = 3.1355 + 137.8080\ 10^{-4} \text{ (NO_2 in metric tons/year).} \quad (3)$$

CO. Ambient concentration of CO in Santiago varies during the year. It is high in the winter months (May to the end of August), at between 10 to 15 parts per million (ppm) in hourly averages. In the summer months (November to February) these average concentrations fall to between 3 and 4 ppm. Air quality standards refer to average concentrations measured over an hour (35 ppm in the United States) or eight hours (9 ppm). To translate the averages in terms of health risk, we use the eight-hour standard. Hence, ambient concentrations of CO in Santiago appear problematic during the winter months but not in the summer months. This probably underestimates the CO problem that may exist in summer, which is concealed by using the weekly averages of hourly measures. Unlike for the other air pollutants, we only have data on CO in Santiago for the base line. Therefore we link emissions and concentrations using the rollback method (elasticity equal to one). Assuming a zero intercept, the dispersion equation is

$$CO \text{ in ppm} = 3.43125\ 10^{-6} \text{ (CO in metric tons/year).} \quad (4)$$

VOCs and O_3. The pollution inventory for Santiago provides VOCs emission levels for the base and control scenario. We use data on O_3 and NO_2 concentrations existing for Santiago and isopleth graphs to recover VOCs concentration. Research has shown that the relationship between O_3 and NO_2 is non-linear. The efficiency of O_3 production decreases with increasing NO_2 concentrations and is sensitive to the VOCs/NO_2 ratio present in the atmosphere. In urban areas where

NO_2 levels are greater than 5 parts per billion (ppb), O_3 formation is more sensitive to VOCs concentrations (National Research Council 1991). We use the following equation to derive changes in O_3 levels (Radian 1995):

$$O_3 = A \text{ x } [(VOCs/NO_2)/(1+(VOCs/NO_2))] + B \text{ x } VOCs + C, \quad (5)$$

with all concentrations expressed in parts per hundred million (pphm). The equation is appropriate for high NO_2 regimes (as would typically be found in urban areas) and is valid for ambient concentrations. We use our linear approximations of emission dispersion for VOCs and NO_2 to obtain the data on levels of ambient concentration of the two pollutants.

We use a typical O_3 isopleth generated with the Empirical Kinetic Modeling Approach (EKMA) from the Environmental Protection Agency's (EPA) Ozone Isopleth Plotting Mechanism (OZIPM-4) model to estimate data on VOCs concentration corresponding to NO_2 and O_3 concentrations. We use the estimated values A=–0.7758, B=0.9525, C=4.9021. To link VOCs emissions and concentration, we use the following equation:

$$VOCs \text{ in pphm} = 21.4233 + 4.4478\ 10\text{-}4 \text{ (VOCs in metric tons/year).} \quad (6)$$

The average ambient level of VOCs in Santiago is estimated at 42.7 pphm for 1992 and corresponds to ambient levels of 17 pphm for O_3 and 6.6 pphm for NO_2.

Lead. For lead we assume a minimum concentration of 0.5 $\mu g/m^3$ and increments based on a rollback model. The slope is such that the 1992 emissions yield a concentration of 1.5 $\mu g/m^3$ (Turner, Weaver and Reale 1993). We have the following model for lead dispersion:

$$Lead \text{ in } mg/m^3 = 0.5 + (\ 1/371.25\ ) \text{ (Lead in metric tons/year).} \quad (7)$$

4. ESTIMATION OF HEALTH ENDPOINTS

The next step involves calculating the physical response of human resources to changes in concentrations of air pollutants. Dose-response functions express the change in incidence of mortality/morbidity induced by changes in pollution concentrations. The response functions used for Santiago come from Ostro (1994), Ostro et al. (1995), The World Bank (1994), and Desvousges et al. (1995). Most of the parameters of the dose-responses come from studies analysing the health impacts of pollutants in Santiago, Chile; Jakarta, Indonesia; and from U.S. data.

Dose-response functions have been found to be readily transferable between countries/studies (Ostro 1994; Desvousges et al. 1995). The use of response functions from other studies assumes similar distribution of baseline factors, such as health status (e.g., incidence of chronic disease), chemical composition of pollutants, occupational exposures, seasonality, time spent outdoors, and general activity.

Most dose-response functions provide point estimates of the response (a change in probability or relative frequency of occurrence) based on a linear, logarithmic or logistic specification. Recovering the probability implies more information (an intercept) and some faith in the ability of point estimates to approximate over a larger range of values. Instead of attempting to recover the probability as a function of the level of the pollutants, we establish a reference point for each dose-response function (by pollutant), which is a concentration standard for the pollutants of interest.

The morbidity and mortality figures are then calculated in deviations with respect to the mortality or morbidity that would prevail when pollution is equal to these standards. The deviation approach allows us to separate the pure effect of changes in pollution on mortality and morbidity from other causes and to abstract from base information on incidence of death, and many morbidity measures.

The baseline information we found on morbidity (e.g., the annual number of emergency rooms visits) appeared to be inconsistent and subject to potential double counting (The World Bank 1995). These shortcomings are the major motivation to follow our approach. We use the following standards: for lead, 0.5 $\mu g/m^3$; for SO_2, 80 $\mu g/m^3$; for NO_2, 5.3 pphm; for CO, 5 pphm; for O_3, 8.17 pphm; and for PM-10, 90 $\mu g/m^3$. The figures on health end-points presented in the results section have to be interpreted as the change in mortality and morbidity with respect to the prevailing standard. Table 1 shows the low, central, and high slopes of the dose-response functions for mortality and morbidity for the six pollutants of interest.

Given the aggregate nature of our model, we do not disaggregate population in Santiago by area satisfying low level of concentrations, which correspond to benign exposure (thresholds). The concentration data we use for the calibration of air pollution in Santiago already reflect population-weighted averages of pollution concentrations. Health impacts are estimated as follows (Ostro 1994):

$$dH_i = b_i \, x \, POP_i \, x \, dA, \tag{8}$$

Table 1. Dose-response function slopes

	Lead (1 μg/m³)			*SO_2 (10 μg/m³)*			*NO_2 (pphm)*		
	Low	*Central*	*High*	*Low*	*Central*	*High*	*Low*	*Central*	*High*
Premature mortality/ 100,000	0	0	0	3.3	3.3	3.3	0	0	0
Premature mortality/ 1 million males age 40-59	200	350	650	0	0	0	0	0	0
RHA/100,000	0	0	0	0	0	0	0	0	0
ERV/100,000	0	0	0	0	0	0	0	0	0
RAD/person	0	0	0	0	0	0	0	0	0
LRI/child	0	0	0	0	0	0	0	0	0
Asthma symptoms/asthmatic	0	0	0	0	0	0	0	0	0
Respiratory symptoms/person	0	0	0	0	0	0	0	0	0
Chronic bronchitis/100,000 age > 25	0	0	0	0	0	0	0	0	0
MRAD/person	0	0	0	0	0	0	0	0	0
Respiratory symptoms/1,000 children	0	0	0	0.1	0.18	0.26	0	0	0
Chest discofort/adult	0	0	0	0.05	0.1	0.15	0	0	0
Respiratory symtom/adult	0	0	0	0	0	0	0.06	0.1	0.14
Eye irritation/adult	0	0	0	0	0	0	0	0	0
Headache/person	0	0	0	0	0	0	0	0	0
IQ decrement[a]	0.975	0.975	0.975	0	0	0	0	0	0
Hypertension/1 million males age >20	44800	72600	97800	0	0	0	0	0	0
Non-fatal heart attacks/ 1 million males age 40-59	180	340	500	0	0	0	0	0	0

Table 1. Extended

	CO (ppm)			*PM-10 (10 µg/m^3)*			*O_3 (pphm)*		
	Low	*Central*	*High*	*Low*	*Central*	*High*	*Low*	*Central*	*High*
Premature mortality/100,000	0	0	0	3.9	4.7	5.7	0.01	0.006	0.01
Premature mortality/ 1 million males age 40-59	0	0	0	0	0	0	0	0	0
RHA/100,000	0	0	0	6.57	12	15.6	3.8	7.7	12
ERV/100,000	0	0	0	128	234	343	0	0	0
RAD/person	0	0	0	0.404	0.575	0.903	0	0	0
LRI/child	0	0	0	0.008	0.016	0.023	0	0	0
Asthma symptoms/asthmatic	0	0	0	0.16	0.33	2.73	0.38	0.68	1.9
Respiratory symptoms/person	0	0	0	0.91	1.83	2.3	0.28	0.55	0.77
Chronic bronchtis/100,000 age > 25	0	0	0	15.5	44	61.7	0	0	0
MRAD/person	0	0	0	0	0	0	0.17	0.34	0.51
Respiratory symptoms/1,000 children	0	0	0	0	0	0	0	0	0
Chest discomfort/adult	0	0	0	0	0	0	0	0	0
Respiratory symptom/adult	0	0	0	0	0	0	0	0	0
Eye irritation/adult	0	0	0	0	0	0	0.23	0.266	0.3
Headache/person	0.01	0.013	0.01	0	0	0	0	0	0
IQ decrement[a]	0	0	0	0	0	0	0	0	0
Hypertension/1 million males age >20	0	0	0	0	0	0	0	0	0
Non-fatal heart attacks 1 million males age 40-59	0	0	0	0	0	0	0	0	0

Note: Low and high estimates represent one standard deviation from central estimate.

[a]Assumes children on average will avoid an IQ loss of 0.975 if they grow up with an ambient lead of 0.5 rather than 1.5 µg/m3.

KEY: RHA = respiratory hospital admissions; LRI = lower respiratory illnes; pphm = parts per hundred million; ERV = emergency room visits; MRAD = minor restricted activity days; ppm = parts per million; RAD restricted activity days; µg/m^3 = micrograms per cubic metre

where dH_i is the change in prevalence of health effect for population i, b_i is the slope of the dose-response function, POP_i is the population at risk of health effect i, and dA is the change in pollution concentration under consideration.

We look at the following incidences: premature mortality due to PM-10, SO_2, and O_3; premature mortality in males of age 40-59 due to lead; respiratory hospital admissions (for PM-10, O_3); emergency room visits (for PM-10); restricted activity days (for PM-10); lower respiratory illness for juvenile population under age 17 (PM-10); asthma symptoms for asthmatic population (for PM-10, O_3); respiratory symptoms (for PM-10, O_3); chronic bronchitis in population age 25 or older (for PM-10); minor restricted activity days (for O_3); respiratory symptoms in juvenile population (for SO_2); chest discomfort in adult population (for SO_2); respiratory symptoms in adult population (for NO_2); eye irritation in adult population (for O_3); number of headaches in adult population (for CO); IQ decrement in juvenile population (for lead); cases of hypertension in adult male population (for lead); and non-fatal heart attacks in male population age 40-59 (for lead).

For each dose-response function we have a central value and a "low" and high" value corresponding to one standard deviation below and above the estimate of the slope of the dose response function (Ostro 1994; Ostro et al. 1995; The World Bank 1994; Desvousges et al. 1995). The dose-response estimates are presented in Table 1. The population data comes from the latest Chilean population census (Banco Central de Chile 1995). Despite the linear nature of the dose-response functions, considerable non-linear effects on health endpoints come from multiplier effects between effluent types (for example, PM-10, NO_2 and SO_2 emissions are highly positively correlated) and from the O_3 estimation.

5. VALUATION OF HEALTH IMPACTS

5.1 Morbidity

We follow the traditional approach of Harrington and Portney (1987), Cropper and Freeman (1991), and Desvousges et al. (1995) in describing the different components entering into the WTP measures with respect to morbidity. Readers interested in a more formal exposé are referred to Freeman (1993). The approach assumes certainty for the states of the world and is based on the household production model. Economic agents maximise their utility, which depends on market good consumption, health, and leisure. Health is "produced" by agents by means of decisions on medical expenses to mitigate the impact of pollution and disease/exposure avoidance for a given environmental quality, say air quality, and given personal physiological endowment such as the agent's

genetic makeup. The approach uses the full income concept of the household model; all endowments and resources of the household/agent are valued at their opportunity cost. Labour income and leisure depend on the health of the individual. Health can be thought of as time in the absence of illness. When pollution increases, health decreases or sick time increases, inducing loss of income and a loss of utility because of a lower health consumption level.

From the first-order conditions to maximise utility given the above constraints and market prices for consumption, avoidance consumption, and mitigating consumption, one can derive optimum decisions or Hicksian demands for the three types of consumption and optimum levels of health and leisure. The derivations also lead to an expenditure function, which depends on the market prices for all types of consumption, the prevailing pollution level, the exogenous physiological endowment, the health level of the individual, and individual's utility level. The WTP to avoid an exogenous increase in pollution is the necessary increase in the agent's expenditure induced by the pollution change such that the agent's utility remains unchanged. This higher pollution affects expenditure related to avoidance consumption decisions, mitigating consumption decisions, the loss of utility due to lower health, and the loss of full income due to increased sick time and decreased leisure time.

Hence, four components are included in the WTP to avoid illness: the opportunity cost of time lost to illness, the opportunity cost of larger avoidance consumption, the cost of medical expenses to mitigate illness, and, finally, the disutility of lower health status. These four components have been estimated and approximated with varying degrees of success. The increase in mitigating expenditure and the lost time can be and have been approximated after the fact. They are referred to as cost of illness. They reflect the observed loss of earnings plus the mitigating medical expenditures. The cost-of-illness measure ignores the value of leisure lost to illness and also abstracts from the opportunity cost of avoidance activities, which are difficult to observe. Hence, they should be considered lower-bound estimates of the WTP for reduced morbidity.

Other estimation methods are used to approximate the WTP for reduced morbidity. Contingent valuation studies use direct surveys of consumers to elicit their WTP to avoid some health risk. Contingent valuation techniques frequently lead to overstatements of WTP when consumers are not familiar with the risk (Viscusi 1993). Contingent valuation, however, provides an estimate of the WTP to reduce morbidity, which includes the four components of WTP discussed above. The individual valuation (cost of illness or WTP based on contingent valuation) generally does not include some additional costs borne by other agents, such as employers or other employees through insurance pools (Freeman 1993). As is typical in environmental economic studies, we use a combination of cost-of-illness and contingent-valuation values to approximate the WTP to reduce the morbidity incidences considered in our study.

Table 2 presents the WTP values and techniques used in this study (cost of illness or contingent valuation). The estimates are expressed in 1992 dollars and account for PPP. The figures correspond to U.S. estimates (ORNL and RFF 1994; Desvousges et al. 1995), which have been rescaled using an adjustment factor based on the ratio of U.S. per capita income to the expected Chilean income per capita for 2010. The estimate of the Chilean income comes from the BAU simulation presented in Chapter 6. The income levels, both for the United States and for Chile, are in 1992 prices to be consistent with the social accounting matrix (SAM) for Chile. The income figures are transformed into purchasing-power-parity dollars (PPP$) using Summers and Heston's (1991) latest estimates from the Penn World Tables 5.6. The conversion to PPP accounts for differences in consumption patterns between countries and measures purchasing power for homogenous goods across countries, allowing for inter-country comparison of real income.[4] The scaling method is a standard procedure in absence of country-specific estimates (Krupnick et al. 1995). For the scaling we assume an elasticity of WTP with respect to income of 2.27 as determined by our mortality valuation function (yet to be described). The elasticity value can be changed if additional knowledge is available on the income response of some of the subcomponents of the WTP (for example, medical expenditures).

The figures in Table 2 reveal substantial variations in the valuation of specific morbidity reductions. The variation is due in part to the different estimation techniques and also to the symptoms and disutility specific to each type of incidence. The figures represent damage per unit for each type of incidence. The estimates of health endpoint impacts of each policy reform scenario are multiplied by the per-unit damage. Their sum constitutes the estimate of total morbidity damage in Santiago attributable to the policy scenario.

5.2 Mortality

The environmental economics literature has established a standard way to value mortality ex ante (see Freeman 1993 and also Viscusi 1993). The approach uses a simple expected utility framework. Income is spent on consumption. Earnings are assumed to increase with riskier occupations. Agents face two sources of risk of death: a "background" risk of death, exogenous to the agent, and an endogenous risk of death, related to the occupational choice of the agent. Individual agents maximise expected utility of aggregate consumption or income. From the differentiation of the expected utility of aggregate consumption, it can be shown that the WTP for a small reduction in the exogenous risk of death reflects the gains in utility induced by the change in the probability of death. The WTP depends on the initial risk of death, the current aggregate consumption of the individual, and the individual's marginal utility of income (or of aggregate consumption).

Table 2. Unit valuation of incidence of morbidity in the U.S. and Chile

	Measure[a]	*U.S. Unit Values*[b] *(1992 PPP$)*	*Chilean Unit Values*[c] *(1992 PPP$)*
Respiratory hospital admission (RHA)	COI	7,134.93	1,212.46
Emergency room visit (ERV)	COI	201.40	34.22
Restricted activity day (RAD)	COI	57.70	9.81
Bronchitis in children (LRI)	COI	149.35	25.38
Asthma attack day	CV	33.94	5.77
Any symptom day (respiratory related)	CV	6.79	1.15
Chronic bronchitis in adults	CV	237,604.84	40,377.05
Minor respiratory restricted activity day (MRRAD)	CV	24.30	4.13
Cough day (or child respiratory symptom day)	CV	5.40	0.92
Chest discomfort case	CV	6.79	1.15
Respiratory symptom day (or phlegm day)	N/A	N/A	N/A
Eye irritation	CV	6.75	1.15
Headache episode (average of mild and severe)	CV	27.19	4.62
IQ decrement (per 1 point IQ loss)	COI	4,726.11	803.13
Hypertension case	Medical Costs Only	455.31	77.37
Non-fatal heart attack	Medical Costs Only	32,020.08	5,441.29

[a] Type of study used to derive health valuation measure: cost-of-illness (COI), contingent valuation (CV) or medical costs only (value of lost wages not included).

[b] Central unit values taken from U.S. studies by ORNL and RFF (1994) and Desvousges et al. (1995). Values from these studies adjusted to 1992 U.S.$ based on CPI (base 1987=100). Note: conversion of U.S.$ to PPP$ not necessary since PPP$ are derived relative to the U.S.$ (i.e., $1 U.S. = $1 PPP).

[c] Estimated using the relative incomes approach (Krupnick et al. 1995), i.e., {U.S. unit value x [(Chilean GDP/capita 2010 in 1992 PPP$)/(U.S. GDP/capital 1992 in 1992 PPP$)]^e where e is the income elasticity of health effect}. Estimate reflects relative income measure of 0.46 based on ratio of Chilean GDP/capita in 2010 to U.S. GDP/capita in 1992, both in 1992 PPP$ terms (i.e., 0.46=$10,636/$23,220). U.S. unit values and GDP per capita are held constant at 1992 PPP$. Conversion assumes income elasticity for health effect of e=2.27. Estimate of e is based on partial elasticity of WTP with respect to income (evaluated at the means) as generated by our mortality valuation model.

From the necessary conditions to maximise the expected utility of aggregate consumption with respect to the occupational choice, a fundamental marginal principle is derived. The WTP for reduced mortality, that is, the monetary value of the utility gain from a smaller risk of occupational death, is equal to the marginal wage income foregone by moving to a safer job. The latter also is almost equal to the WTP for a small change in the exogenous risk of death, if the two risks, background and occupational, are small or equal (see Freeman 1993, pp. 328-9, for a formal derivation of this result). The fundamental approximation motivates the use of information on wage-risk trade-offs to estimate the WTP for small reductions in exogenous mortality related to pollution.

To estimate the WTP for reduced mortality, we conduct a meta-analysis of the numerous existing wage-risk studies in industrialised countries, from which we obtain 33 data points on wage differentials and occupational risk. These studies provide estimates of compensation differentials across occupations for different mortality risk levels. The studies use different methodologies and cover different time periods, countries, and risk levels. The differential is simply the premium received for engaging in a riskier occupation. Several concerns are addressed. Because the studies use different specifications linking wage and risk and rely on data of heterogeneous quality, data are likely to violate assumptions underlying statistical inference[5] and simple techniques such as ordinary least-squares (OLS) regression. To remedy the shortcomings of the data we use regression diagnostic techniques to identify the points that are influential, that is, the points that have a strong role in determining the value of estimated coefficients[6] because of undue influence.

Another concern is the fact that we use data from industrialised countries with high income—an older and better-educated population—to predict "out of sample," that is, in lower-income countries characterised by a younger and less-educated population. A further out-of-sample dimension is that environmental risk tends to be smaller than the occupational risk of the labour market studies. It is well known in statistics that the accuracy of prediction decreases quickly for data points outside of the range of the data used for the estimation. Hence, one of our criteria to select a preferred model, among the many models we estimated, is the way the model predicts observations corresponding to low-income, risk, and compensating differentials such as is the case for our Chilean investigation.[7]

A third concern related to the previous one has to do with the functional form used to estimate the relationship between the compensating differential and its explanatory variables. Because we intend to use the estimated coefficients to predict outside of the sample, we want to be sure that the chosen functional form is flexible enough (has enough "curvature") to fit the data over the whole range of the observed data. For example, a linear model may only predict well around the mean of the data set.

The last concern is the choice of the explanatory variables. The theory underlying the estimation clearly links the compensating differential to risk and

initial income (the utility of aggregate consumption). Besides the two fundamental explanatory variables, other determinants, such as age, education (a proxy for human capital and information on risk), and gender, may influence the compensating differentials. We try different combinations of variables, all of which include a set of core variables (income, risk, education, and age). These variables are the most likely to distinguish developing economies' situations from those of the industrialised countries. In summary, we estimate the following equation for the compensating differential, CD:

$$CD = CD(income, risk, age, education, other\ noncore\ determinants), \quad (9)$$

where income is annual wages, risk is the number of occupational deaths per 10,000, education is the number of years of education, and other noncore determinants are variables such as gender and nonwage compensation (Bowland 1997; Bowland and Beghin 2001).[8]

We ran over fifty specifications. In Table 3, we report the results for eight specifications of special interest because they represent the canonical forms used by economists (linear, quadratic, semi-log, double-log, quadratic in logs, and translog). The table describes the functional form and associated elasticities of the compensating differential with respect to the four core determinants evaluated at the mean of the variables for each model. Two of the models (6 and 14) include additional variables on gender, nonwage compensation, and union membership. We ran these specifications using ordinary least squares and robust regression.[9] Robust regression down-weighs influential data points.

As shown in Table 3, several strong results emerge from the econometric modelling. Compensating differential is positively related to risk, but the response is inelastic (median elasticity value of 0.25 for the 16 estimated values). The compensating variable responds positively to income change, and the elasticity value is large, with a median value of 1.77. These results hold for most models. The positive and generally elastic response of the compensating differential to the average education of the population investigated is another systematic result. The elasticity value has a moderate range, with the median of estimated values equal to 1.4. Results pertaining to the age variable show a negative response, but the results are not significant in either OLS or robust runs and exhibit substantial variations in magnitude of the elasticity.

Based on our criteria of mean-square prediction error, three specifications (13-15) perform the best. Using the results of the robust estimation of the eight specifications, we predict the WTP to avoid the mortality increase occurring between 1992 and 2010 in the BAU scenario. We value the WTP at the 2010 income expressed in 1992 PPP$. Age and education are the current values for Chile, which we use as best guesses of the future 2010 levels. We follow the same step for the WTP to avoid the mortality associated with the pollution resulting in 2010 under the environmental policy reform decreasing PM-10 by

Table 3. Partial elasticities (at the means) for 8 OLS and 8 robust CD models

		$e_{CD,RISK}$	$e_{CD,INC}$	$e_{CD,AGE}$	$e_{CD,EDUC}$
	OLS				
(1)	$CD = \beta_0 + \Sigma^4_{i=1} \beta_i X_i$	0.236*	1.682*	-0.399	1.194
(2)	$CD = \beta_0 + \Sigma^4_{i=1} \beta_i X_i + \Sigma^4_{i=1} \gamma_i X_i^2$	0.046	1.970	-2.089	1.963
(3)	$CD = \beta_0 + \Sigma^7_{i=1} \beta_i X_i$	0.197*	1.590*	-0.457	1.869
(4)	$CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i$	0.308*	1.388*	0.098	0.736
(5)	$\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i$	0.294*	2.098*	-0.850	1.608
(6)	$\ln CD = \beta_0 + \Sigma^5_{i=1} \beta_i \ln X_i + \Sigma^7_{i=6} \gamma_i X_i$	0.300*	2.256*	-0.774	2.598*
(7)	$\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i + \Sigma^4_{i=1} \gamma_i \ln X_i^2$	0.475*	1.849	0.505	1.030
(8)	$\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i + \frac{1}{2}\Sigma^4_{i=1}\Sigma^4_{j=1} \gamma_{ij} \ln X_i \ln X_j$	0.133*†	1.408	-3.250	0.739
	ROBUST				
(9)	$CD = \beta_0 + \Sigma^4_{i=1} \beta_i X_i$	0.199*	1.657*	-0.527	1.145
(10)	$CD = \beta_0 + \Sigma^4_{i=1} \beta_i X_i + \Sigma^4_{i=1} \gamma_i X_i^2$	0.146	1.925	-2.215	1.766
(11)	$CD = \beta_0 + \Sigma^7_{i=1} \beta_i X_i$	0.164**	1.556*	-0.504	1.893*
(12)	$CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i$	0.272*	1.378*	-0.107	0.682
(13)	$\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i$	0.307*	2.093*	-0.863	1.905*
(14)	$\ln CD = \beta_0 + \Sigma^5_{i=1} \beta_i \ln X_i + \Sigma^7_{i=6} \gamma_i X_i$	0.306*	2.269*	-0.802	2.578*
(15)	$\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i + \Sigma^4_{i=1} \gamma_i \ln X_i^2$	0.522*	1.901	-0.186	0.989
(16)	$\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i + \frac{1}{2}\Sigma^4_{i=1}\Sigma^4_{j=1} \gamma_{ij} \ln X_i \ln X_j$	0.149*†	1.520	-4.080	0.733
	Med	0.254	1.765	-0.651	1.401
	Min	0.046	1.378	-4.080	0.682
	Max	0.522	2.269	0.505	2.598
	Std. Dev.	0.123	0.303	1.249	0.644

Source: Bowland 1997; Bowland and Beghin 2001.

*Partial elasticity significant at p-value≤0.05 according to t-statistics for betas.

**Partial elasticity significant at p-value=0.06 according to t-statistics for betas.

†Regression coefficient on squared risk term was significant at p-value=0.34. However, all other regression coefficients on risk interaction terms were significant at p-value≤0.05.

25 per cent. The WTP estimates are shown in Table 4. The table also shows the

difference in WTP between the two scenarios (BAU minus PM-10 tax policy reform). The difference expresses the change in the WTP for a reduction in mortality induced by the environmental tax on PM-10. The table shows each of the three values computed for high and low estimates of the risk of death. The estimates correspond to the high and low levels of the dose-response functions. The table also shows the dramatic variation in the estimated WTP in the given scenario. The variation primarily is caused by the change in specifications. Specification 16, the translog, provides nonsensical economic results because the WTP increases whenever the mortality decreases (e.g., holding risk changes between scenarios constant, an increase from low to high risk under BAU decreases WTP using the translog specification). This variation is a strong motivation not to select this functional form.

Differences can be taken between the two measures of the WTP to reduce mortality, as implied by the BAU scenario and the environmental tax scenario. The difference provides an estimate of the partial effect of the decreased mortality, induced by the tax on PM-10, on the WTP to avoid mortality. As shown in the third column of Table 4, the interspecification variation is much reduced. This is due to the fact that the influence of the intercept, age, education, and other variables has been reduced. The partial effect captures the risk change and the moderate income change between the two scenarios (BAU and the tax). We make use of the differences later when we look at damages under a few policy scenarios. Our preferred functional form is the fourteenth because it ranks well in terms of prediction. Further, the double-log specification yields values of WTP that are close to the median of the estimates.

The last step to value the damage associated with mortality is to multiply the estimated individual WTP (or CD_i) for reduced mortality by the population at risk ($Population_i$) and then divide the sum of WTP by the expected reduction in number of deaths for that population ($DRisk_i$). The number obtained by the transformations is called the value of a statistical life (VSL). Formally it is presented as follows:

$$VSL = CD_i(DRisk_i, Income, Other\ non\text{-}income\ determinants)\ x\ Population_i / DRisk_i, \quad (10)$$

where CD_i is a function of risk changes, level of income, and other nonincome determinants such as education for individuals of the ith population at risk; and $Population_i$ is the number of individuals in the ith population at risk. VSL expresses a human group's ex ante aggregate WTP to reduce mortality. As shown in Table 5, our computations yield an estimated value of a statistical life in Santiago of around $550,000 1992 PPP$ under BAU. This is at the lowest end of the values reported in the data set used to estimate the compensating differential. VSL varies for each policy scenario because risk and income levels vary by scenario and thus alter the value of CD between scenarios.

Table 4: Estimated individual willingness to pay in Santiago to forego changes in risk of mortality due to PM-10 pollution in 2010 by robust model specification and policy (in 1992 PPP$ at 2010 income levels)

		Individual WTP		
		(1)	*(2)*	*(3)*
Robust Model Specification	*DRisk*[a]	*BAU*	*PM-10 Tax Policy Reform*	*[(2) - (1)]*
(9) $CD = \beta_0 + \Sigma^4_{i=1} \beta_i X_i$	low	278	192	-86
	high	374	249	-124
(10) $CD = \beta_0 + \Sigma^4_{i=1} \beta_i X_i + \Sigma^4_{i=1} \gamma_i X_i^2$	low	794	731	-63
	high	889	773	-116
(11) $CD = \beta_0 + \Sigma^7_{i=1} \beta_i X_i$	low	279	208	-71
	high	358	255	-103
(12) $CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i$	low	227	116	-111
	high	305	195	-111
(13) $\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i$	low	229	194	-35
	high	257	218	-39
(14) $\ln CD = \beta_0 + \Sigma^5_{i=1} \beta_i \ln X_i + \Sigma^7_{i=6} \gamma_i X_i$	low	272	231	-41
	high	306	259	-47
(15) $\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i + \Sigma^4_{i=1} \gamma_i \ln X_i^2$	low	131	115	-16
	high	131	127	-4
(16) $\ln CD = \beta_0 + \Sigma^4_{i=1} \beta_i \ln X_i + \frac{1}{2}\Sigma^4_{i=1}\Sigma^4_{j=1} \gamma_{ij} \ln X_i \ln X_j$	low	161	295	134
	high	98	189	91

Source: Bowland.

Note: Column headings are as follows:

(1) Estimated individual WTP in Santiago to avoid changes in the risk of death associated with increased pollution over 1992 levels under BAU.

(2) Estimated individual WTP in Santiago to avoid changes in the risk of death associated with increased pollution over 1992 levels under a PM-10 tax reform policy (decreasing PM-10 emissions by 25 per cent relative to BAU in 2010).

(3) Change in estimated individual WTP under PM-10 tax reform relative to BAU [i.e., columns (2) - (1)].

[a]The change in risk of death due to PM-10 levels varies from low to high because of different slope estimates from the dose-response functions (see Table 1).

Table 5: Estimated premature mortality and implied value of a statistical life for Santiago in 2010 by policy scenario

Policy Scenario	*Premature Mortality[a] (deaths)*	*Total WTP to Avoid Premature Mortality[b] (billions 1992 PPP$)*	*Implied VSL[c] (1992 PPP$)*
BAU (business as usual)	4321	2.38	550,533
ENV1 (tax on lead)	4295	2.35	547,630
ENV2 (tax on SO_2)	2787	2.07	744,240
ENV3 (tax on PM-10)	2653	2.04	768,347
ENV4 (tax on NO_2)	2797	2.08	742,409
NAFTA (NAFTA integration)	4485	2.48	553,513
FTLIB (unilateral FT liberalisation)	5620	2.92	518,746
ENAFTA1 (ENV1 + NAFTA)	4447	2.46	552,953
ENAFTA2 (ENV2 + NAFTA)	2772	2.14	771,094
ENAFTA3 (ENV3 + NAFTA)	2625	2.10	798,953
ENAFTA4 (ENV4 + NAFTA)	2771	2.14	771,198
EFTLIB1 (ENV1 + FTLIB)	5626	2.89	512,882
EFTLIB2 (ENV2 + FTLIB)	2858	2.35	824,013
EFTLIB3 (ENV3 + FTLIB)	2682	2.30	857,703
EFTLIB4 (ENV4 + FTLIB)	2866	2.36	822,320

[a] Central estimate of premature deaths in 2010 due to increases in air pollution (lead, SO_2, PM-10, O_3) over 1992 levels by policy scenario.
[b] Damages from premature mortality estimated as the sum of WTP by citizens of Santiago to avoid changes in the probability of premature death associated with increased pollution over 1992 levels.
[c] Implied value of a statistical life (VSL) estimated as [(total damages ÷ (deaths)] x 1,000,000,000.

6. AN APPLICATION TO HEALTH DAMAGES IN SANTIAGO INDUCED BY POLICY REFORMS

Table 6 reports pollution damages for some of the policy reforms considered in Chapter 6. The pollution damages are valued at the income corresponding to the real gross domestic product (GDP) level implied by each policy scenario, transformed into 1992 PPP$. The table shows the consequences of the reforms on GDP and health damages in Santiago. Both GDP and health dam-

Table 6. Total Chilean gross domestic product and Santiago health damages in 2010 by policy scenario relative to business as usual

	Total Chilean GDP in 2010			
Policy Scenario	*(1) Billions (1992 Pesos)*	*(2) Billions (1992 PPP$)*	*(3) D w.r.t. BAU (1992 PPP$)*	*(4) D w.r.t. BAU (%)*
BAU (business as usual)	33,230.8	191.3	0	0
ENV1 (tax on lead)	33,131.1	190.8	-0.57	-0.3
ENV2 (tax on SO_2)	33,164.3	190.9	-0.38	-0.2
ENV3 (tax on PM-10)	33,131.1	190.8	-0.57	-0.3
ENV4 (tax on NO_2)	33,164.3	190.9	-0.38	-0.2
NAFTA (NAFTA integration)	33,696.0	194.0	2.68	1.4
FTLIB (unilateral FT liberalisation)	35,091.7	202.0	10.71	5.6
ENAFTA1 (ENV1 + NAFTA)	33,629.6	193.6	2.30	1.2
ENAFTA2 (ENV2 + NAFTA)	33,629.6	193.6	2.30	1.2
ENAFTA3 (ENV3 + NAFTA)	33,596.3	193.4	2.10	1.1
ENAFTA4 (ENV4 + NAFTA)	33,629.6	193.6	2.30	1.2
EFTLIB1 (ENV1 + FTLIB)	34,992.0	201.5	10.14	5.3
EFTLIB2 (ENV2 + FTLIB)	34,958.8	201.3	9.95	5.2
EFTLIB3 (ENV3 + FTLIB)	34,892.3	200.9	9.57	5.0
EFTLIB4 (ENV4 + FTLIB)	34,958.8	201.3	9.95	5.2

Note: Column headings are as follows: (1) Total real Chilean GDP in 2010 denominated in billions of 1992 pesos as given by CGE model; (2) Total real Chilean GDP in 2010 denominated in billions of 1992 PPP$ (purchasing power parity international prices relative to the U.S. dollar). Conversion of pesos to PPP$ based on Summers and Heston's (1991) estimates of 1992 PPP for Chile found in their Penn World Tables (Mark 5.6); (3) Change in total Chilean GDP by policy scenario relative to BAU in 2010 (in PPP$ terms); (4) Change in total Chilean GDP by policy scenario relative to BAU in 2010 (in per centage terms).

Table 6: Extended

	Total Health Damages in 2010			*D Net Welfare*
	(5)	*(6)*	*(7)*	*(8)*
Policy Scenario	*Billions (1992 PPP$)*	*Dw.r.t BAU (1992 PPP$)*	*Dw.r.t. BAU (%)*	*[(3) - (6)]*
BAU (business as usual)	5.88	0	0	0
ENV1 (tax on lead)	5.31	-0.58	-9.80	0.00
ENV2 (tax on SO_2)	5.19	-0.69	-11.71	0.31
ENV3 (tax on PM-10)	4.89	-0.99	-16.87	0.42
ENV4 (tax on NO_2)	5.19	-0.69	-11.72	0.31
NAFTA (NAFTA integration)	6.12	0.24	4.07	2.44
FTLIB (unilateral FT liberalisation)	7.61	1.73	29.40	8.99
ENAFTA1 (ENV1 + NAFTA)	5.58	-0.30	-5.14	2.60
ENAFTA2 (ENV2 + NAFTA)	5.34	-0.55	-9.27	2.84
ENAFTA3 (ENV3 + NAFTA)	4.99	-0.89	-15.14	2.99
ENAFTA4 (ENV4 + NAFTA)	5.33	-0.55	-9.33	2.84
EFTLIB1 (ENV1 + FTLIB)	6.69	0.81	13.71	9.33
EFTLIB2 (ENV2 + FTLIB)	6.27	0.39	6.64	9.56
EFTLIB3 (ENV3 + FTLIB)	5.77	-0.11	-1.93	9.68
EFTLIB4 (ENV4 + FTLIB)	6.27	0.39	6.61	9.56

Note: Column headings are as follows: (5) Total health damages in 2010 for Santiago denominated in billions of 1992 PPP$. Total health damages represent sum of premature mortality and incidence of morbidity for citizens of Santiago in 2010; (6) Change in total health damages for Santiago by policy scenario relative to BAU in 2010 (in PPP$ terms); (7) Change in total health damages for Santiago by policy scenario relative to BAU in 2010 (in percentage terms); (8) Change in net welfare for Chile by policy scenario relative to BAU in 2010 (in PPP$ terms). Represents the difference of changes in total Chilean GDP and changes in total health damages for Santiago by policy scenario relative to BAU in 2010.

ages are expressed first in levels, then in differences from their BAU levels, and finally in per cent changes from their BAU levels. Health damages are considerable. For example, FTLIB (unilateral free trade liberalisation) increases health damages by 29 per cent with respect to BAU. This is equivalent to 16 per cent of the GDP gains with respect to BAU. Under the NAFTA scenario, health damages slightly increase by 4 per cent with respect to BAU. The moderate increase in damages in this case is due to the limited increase in urban air pollution induced by joining NAFTA.

A second important fact emerging from the figures in Table 6 is that environmental taxes on air pollution generate important reductions in health damages with little foregone growth. The sum of the two effects is positive, corresponding to a welfare gain. This is an approximation of "true welfare" gain. True welfare changes are difficult to compute in a CGE model framework when nonmarket goods are at issue (Sadoulet and de Janvry 1995). For example, assumptions regarding separability in consumer preferences implies that nonmarket environmental goods will have no effect on marginal rates of substitution among other market goods and thus have no feedback effects on relative prices (see Espinosa 1996; and Espinosa and Smith 1995 for details). As a second-best approximation, we estimate changes in net welfare as the difference between dollar GDP benefits and dollar health damages for each scenario relative to BAU after solving the CGE model (see column 3 through column 6 in Table 6).[10]

Because the health damages refer to urban population in Santiago and not the whole Chilean population, the figure in the table provides a lower bound of the damage reduction induced by environmental taxes. The figures pertaining to the coordinated reform (air pollution taxes plus unilateral free trade) show less beneficial effects of the environmental taxes relative to BAU due to important substitution effects across pollutants, which are exacerbated under free trade (see column 6). For instance, the tax on PM-10 reduces mortality but also increases morbidity induced by higher emissions of other pollutants. Hence, higher damages are associated with the latter. Overall health damages still decrease compared to the BAU damages, and the overall benefits (GDP growth plus reduced health damages) exceed the net benefits associated with free trade alone.

7. CONCLUDING COMMENTS

We showed how pollution emissions were transformed into ambient pollution and health impacts on urban population and the resulting health damages. We explained how we had to rely on indirect information to value morbidity and mortality, because no such data are available for developing economies such as

Chile. The morbidity valuation was straightforward and essentially consisted of scaling damage estimates per incidence by the ratio of Chile's income per capita over U.S. income per capita.

The estimation of mortality damages was more sophisticated. It exhausted the extensive information existing on mortality valuation in industrialised countries and systematically accounted for differences in income, risk of death, age, education, and other socio-economic variables, between Chile and the industrialised countries. We found that the estimate of WTP for reduced mortality is very sensitive to the chosen functional form. However, most estimated specifications exhibit an inelastic response to changes in risk of death but exhibit an elastic response to income changes.

The valuation of the health consequences of policy reforms shows that abstracting from health damages generated by air pollution seriously biases the assessment of policy reform. We showed that considerable net benefits were associated with abatement of urban air pollution, despite the lower GDP resulting from environmental taxes. Conversely, income growth induced by free trade alone is "oversold" as welfare improving to the extent that substantial health damages are not accounted for.

NOTES

[1] Without implicating them, we thank Sebastien Dessus, Raul O'Ryan, David Roland-Holst, Kerry Smith, Dominique van der Mensbrugghe, and participants at the AAEA Meetings, Camp Resources IV, North Carolina State University, Universidad de Chile and Conoma for comments and suggestions.

[2] The only exceptions are the current work by Liu and co-authors, and by A. Alberini, M. Cropper, and associates in Taiwan and India (see Liu and Hammitt 1999; Liu et al. 1997; and Alberini et al. 1997).

[3] Figures reported in chapter 6 are slightly lower. These figures are based on an updated pollution inventory for Santiago, which showed lower ambient pollution levles. See note 1 page 164.

[4] Purchasing power parity conversion is typically used by health economists when analysing cross-country health expenditure data because exchange rate conversion is insensitive to nontradables such as health care (Gerdtham and Jonsson 1991).

[5] Namely, non-normality may be present in the residuals, which will invalidate any inference on the regressions estimates. Further, Gauss-Markov assumptions are likely to be violated since different research approaches have been used to generate the data on compensating differentials and risk.

[6] Data points are influential because of high leverage and/or large standardised residuals. We compute DFFITS and DFBETAS statistics for each specification (see Besley, Kuh, and Welsh 1980 for definitions of these statistics). Two observations were outliers and overly influential in many specifications.

[7] We use mean-square error of prediction measures for the bottom 5 and 10 observations when the data is sorted by increasing order of risk, income, and compensating differentials.

[8] Data is available from the authors.

[9] For our robust regressions we use the iteratively reweighted least squares (IRLS) method, which lowers the importance of influential data points for which residuals exceed a critical level. We use Huber and Bisquare weights. The table presents the results for the Huber weights. Huber weights are "descending" weights, which do not exclude any data points. The Bisquare weights procedure excludes extreme outliers.

[10] Although we assume separability, we somewhat avoid the stronger restriction of fixed "price" health effects for damages by allowing changes in income to affect morbidity valuation and changes in risk and income to affect the VSL.

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